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Title
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Permalink
https://escholarship.org/uc/item/0s3477fm

Journal
Stroke, 35(1)

ISSN
0039-2499

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Publication Date
2004

DOI
10.1161/01.str.0000105387.38892.98

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Editorial Comment

Spasticity After Stroke: What's the Catch?

Motor deficits are the most common impairment acutely after stroke and persist in nearly half of all patients.[1,2] Although much focus is on hemiparesis in this setting, injury to the motor system does not produce a homogenous clinical syndrome. Instead, weakness may be accompanied by other negative findings such as slowness and fatigue and by positive findings such as synkinesia and spasticity.

Spasticity is a state of increased tone with exaggerated reflexes resulting from upper motor neuron injury. It is a condition of many contrasts. Reduced activity in one area, the descending motor tracts, results in increased activity in another area, the skeletal muscles. Spasticity is common across neurological conditions, yet accurate measurement is difficult. It is associated with weakness, yet its maintenance is critical to function in some patients. Importantly, spasticity remains a key dividing point among major schools of physiotherapy, with some aiming to inhibit[3] and others aiming to encourage[4] spasticity and its accompanying motor abnormalities. The medical system expends substantial resources to reduce spasticity with methods that include botulinum toxin injection, intrathecal medication, oral pharmacological agents, and physical/occupational therapy. Yet, limited information is available on its prevalence and significance after stroke. Indeed, in a recent review, Barnes[5] noted the limited availability of quality data on the prevalence of spasticity after stroke.

Some of those data are now available. Sommerfeld et al[6] studied consecutive patients with a first stroke over a 10-month period. Among 95 patients assessed a mean of 5 days after stroke, 21% had spasticity and 81% had hemiparesis. Three months later, 19% were spastic and 67% were hemiparetic. Of note, only 28% of the hemiparetic patients had spasticity. A weakness of the study is that the authors provided limited detail as to precisely which muscles were affected by spasticity. Values for spasticity prevalence in this study are not likely an underestimation, because any measurable increase in tone was considered to constitute spasticity. Study results suggest that although spasticity is associated with greater deficits

and disability, it is present in a minority of stroke patients and in a minority of hemiparetic stroke patients.

The indications for reducing spasticity after stroke remain a topic of ongoing investigation. A compelling argument can be made for treating spasticity after stroke in certain specific instances, eg, when the goal is to prevent an incipient contracture or to reduce a regional pain syndrome such as that associated with a hemiplegic shoulder.[7–9] However, improvement in overall coordinated movement or in disability after stroke as a general response to reduction of spasticity remains to be firmly established.[10] Indeed, treatments targeting spasticity have often had difficulty demonstrating functional benefit.[11] Dobkin[8] recently noted in this context, "With the exception of lessening painful or disruptive spasms and dystonic postures, drugs in general do not decrease impairments or lessen disabilities."

A range of additional studies is needed to refine guidelines for treating spasticity after stroke. As with so many aspects of stroke, response to spasticity-related therapy may be maximum in a subset of patients or may be realized in performance of a subset of motor tasks.[12] The effects of such therapy may be best measured not by general neurological outcome scales but rather by the use of end points most relevant to effects of spasticity.[13] Newer instrumentation-based methods might also improve measurement of spasticity.[14,15] Clinical trials may further clarify the utility of specific approaches to reduce spasticity. The study by Sommerfeld et al,[6] by providing quality data on the prevalence and functional significance of spasticity after stroke, is an important step.

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References

1. Gresham GE, Duncan PW, Stason WB, Adams HP, Adelman AM, Alexander DN, Bishop DS, Diller L, Donaldson NE, Granger CV, et al. *Post-Stroke Rehabilitation*. Rockville, Md: US Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research; 1995.
2. Rathore SS, Hinn AR, Cooper LS, Tyroler HA, Rosamond WD. Characterization of incident stroke signs and symptoms: findings from the Atherosclerosis Risk in Communities Study. *Stroke*. 2002;33:2718–2721.
3. Bobath B. *Adult Hemiplegia: Evaluation and Treatment*. Oxford, UK: Butterworth-Heinemann; 1990.
4. Brunnstrom S. *Motor Behavior of Adult Patients With Hemiplegia: Movement Therapy in Hemiplegia*. New York, NY: Harper & Row; 1970.
5. Barnes MP. Medical management of spasticity in stroke. *Age Ageing*. 2001;30(suppl 1):13–16.
6. Sommerfeld DK, Eek E U-B, Svensson A-K, Holmqvist LW, von Arbin MH. Spasticity after stroke: its occurrence and association with motor impairments and activity limitations. *Stroke*. 2004;35:134–140.
7. Barnes MP, Ward AB. *Textbook of Rehabilitation Medicine*. Oxford, UK: Oxford University Press; 2000.
8. Dobkin BH. *The Clinical Science of Neurologic Rehabilitation*. New York, NY: Oxford University Press; 2003.
9. Teasell RW, Heitzner JD. The painful hemiplegic shoulder. *Phys Med Rehabil State Art Rev*. 1998;12:489–500.
10. Landau WM. Botulinum toxin for spasticity after stroke. *N Engl J Med*. 2003;348:258–259; author reply 258–259.
11. Bhakta BB. Management of spasticity in stroke. *Br Med Bull*. 2000;56:476–485.
12. Hurvitz EA, Conti GE, Brown SH. Changes in movement characteristics of the spastic upper extremity after botulinum toxin injection. *Arch Phys Med Rehabil*. 2003;84:444–454.
13. Brashear A, Gordon MF, Elovic E, Kassicieh VD, Marciniak C, Do M, Lee CH, Jenkins S, Turkel C. Intramuscular injection of botulinum toxin for the treatment of wrist and finger spasticity after a stroke. *N Engl J Med*. 2002;347:395–400.
14. Sehgal N, McGuire JR. Beyond Ashworth. Electrophysiologic quantification of spasticity. *Phys Med Rehabil Clin N Am*. 1998;9:949–979, ix.
15. Johnson GR. Outcome measures of spasticity. *Eur J Neurol*. 2002;9(suppl 1):10–16; discussion 53–61.